

## THE CLAIMS

1. (currently amended) A method for reducing vascular hyperreactivity in vascular muscle cells comprising exposing the vascular muscle cells to an effective amount of a selective estrogen beta receptor agonist that has a higher relative selectivity than does genistein for estrogen receptor beta compared to estrogen receptor alpha is selected from the group consisting of 5 $\alpha$ -androstane-3 $\beta$ ,17 $\beta$ -diol, a derivative of 5 $\alpha$ -androstane-3 $\beta$ ,17 $\beta$ -diol that has estrogen beta receptor agonist activity, epiestriol, and diarylpropionitrile.

2. (previously presented) The method of claim 1 wherein the vascular hyperreactivity is manifested by coronary arterial vasospasm.

3. (previously presented) The method of claim 1 wherein the vascular hyperreactivity is manifested by hyperreactivity of peripheral arteries.

4. (original) The method of claim 1 wherein the estrogen beta receptor agonist is 5 $\alpha$ -androstane-3 $\beta$ ,17 $\beta$ -diol.

5. (original) The method of claim 1 wherein the estrogen beta receptor agonist is a derivative of 5 $\alpha$ -androstane-3 $\beta$ ,17 $\beta$ -diol that has estrogen beta receptor agonist activity.

6. (original) The method of claim 5 wherein the derivative of 5 $\alpha$ -androstane-3 $\beta$ ,17 $\beta$ -diol is selected from the group consisting of 5 $\alpha$ -androstane-3 $\beta$ ,17 $\beta$ -diol-3 hemisuccinate,

5 $\alpha$ -androstane-3 $\beta$ ,17 $\beta$ -diol-17-sulphate sodium salt, 5 $\alpha$ -androstane-3 $\beta$ ,17 $\beta$ -diol-3-acetate, 5 $\alpha$ -androstane-3 $\beta$ ,17 $\beta$ -diol-17-acetate, 5 $\alpha$ -androstane-3 $\beta$ ,17 $\beta$ -diol-diacetate, 5 $\alpha$ -androstane-3 $\beta$ ,17 $\beta$ -diol-dibenzoate, 5 $\alpha$ -androstane-3 $\beta$ ,17 $\beta$ -diol-dihemisuccinate, 5 $\alpha$ -androstane-3 $\beta$ ,17 $\beta$ -diol-dipropionate, and 5 $\alpha$ -androstane-3 $\beta$ ,17 $\beta$ -diol-17-hexahydrobenzoate.

7. (original) The method of claim 1 wherein the estrogen beta receptor agonist is epiestriol.

8. (previously presented) The method of claim 1 wherein the estrogen beta receptor agonist is diarylpropionitrile.

9. (previously presented) The method of claim 4 wherein the 5 $\alpha$ -androstane-3 $\beta$ ,17 $\beta$ -diol is administered to a patient and the amount of 5 $\alpha$ -androstane-3 $\beta$ ,17 $\beta$ -diol that is administered is sufficient to obtain a serum concentration of between 30 and 3000 pg/ml.

10. (original) The method of claim 9 wherein the amount of 5 $\alpha$ -androstane-3 $\beta$ ,17 $\beta$ -diol that is administered is sufficient to obtain a serum concentration of between 30 and 300 pg/ml.

11. (previously presented) The method of claim 1 wherein the estrogen beta receptor agonist is administered to a patient in concert with a hormone replacement therapy.

12. (original) The method of claim 11 wherein the hormone replacement therapy is selected from the group consisting of estrogen, androgen, and progestin therapy.

13. (previously presented) The method of claim 1 wherein the exposure of the vascular muscle cells to the estrogen beta receptor agonist is by administering the estrogen beta receptor agonist by topical application to skin of a patient.

14. (original) The method of claim 13 wherein the estrogen beta receptor agonist is in a topical preparation selected from the group consisting of a liquid, cream, gel, lotion, ointment, and transdermal patch.

15. (previously presented) The method of claim 1 wherein the exposure of the vascular muscle cells to the estrogen beta receptor agonist is by other than topical administration to skin.

16. (original) The method of claim 15 wherein the exposure is by oral, rectal, vaginal, topical, sublingual, nasal, intradermal, inhalation, or sustained implant administration routes.

17-23. (canceled)